

Honeywell Hydrofluoric Acid

Recommended Medical Treatment for Hydrofluoric Acid Exposure

Honeywell

Sustainable Opportunity Policy

Honeywell's Commitment to Health, Safety and the Environment

By integrating health, safety and environmental considerations into all aspects of our business, we protect our employees, our communities and the environment, achieve sustainable growth and accelerated productivity, drive compliance with all applicable regulations and develop technologies that expand the sustainable capacity of our world. Our health, safety and environmental management systems reflect our values and help us meet our business objectives.

- We protect the safety and health of our employees, and minimize the environmental footprint of our operations through efforts to prevent illness, injury and pollution.
We actively promote and develop opportunities for expanding sustainable capacity by increasing fuel efficiency, improving security and safety, and reducing emissions of harmful pollutants.
We are committed to compliance with all of our health, safety, environmental and legal requirements everywhere we operate.
Our commitment to health, safety and the environment is an integral aspect of our design of products, processes and services, and of the lifecycle management of our products.
Our management systems apply a global standard that provides protection of both human health and the environment during normal and emergency situations.
We identify, control and endeavor to reduce emissions, waste and inefficient use of resources and energy.
We are open with stakeholders and work within our communities to advance laws, regulation and practices that safeguard the public.
We abide by the company's own strict standards in cases where local laws are less stringent.
Our senior leadership and individual employees are accountable for their role in meeting our commitments.
We measure and periodically review our progress and strive for continuous improvement.

These are our commitments to health, safety, and the environment, and to creating Sustainable Opportunity everywhere we operate.

[Signature of Dave Cote]

Dave Cote
Chairman and CEO
1/1/2010

[Signature of Andreas Kramvis]

Andreas Kramvis
President and CEO
Performance Materials and Technologies
12/16/2011

Honeywell Performance Materials and Technologies
Responsible Care® Commitment

At Honeywell Performance Materials and Technologies, we are committed to the safety of our employees, the quality of our products, and being responsible stewards for the protection of our environment, the communities in which we operate, and our customers. We are a member company of the American Chemistry Council, and Responsible Care® is the foundation for sustainability in our business. Our Responsible Care® Management System is used to support our full commitment to comply with legal and other Health, Safety and Environmental (HS&E) requirements to which we subscribe and to drive continual improvement in these areas.

We achieve global operational excellence and reliability through the integration of Responsible Care® principles into the way we operate and work with our commercial partners - from our contractors and other suppliers to our customers. We conduct thorough product risk assessments prior to commercialization and we apply necessary resources and best practices in the development and handling of chemical products and materials. We promote process safety through our management systems for the design, construction, installation and maintenance of our facilities. We use quantitative and qualitative methodologies to evaluate enterprise risk and develop risk mitigation measures. As we strive toward environmental excellence and the prevention of pollution, we protect individual and public safety by manufacturing, transporting and storing our materials in a secure manner.

We invest in and improve the compliance processes for our products, processes and services using quantifiable goals to drive sustained safety and environmental excellence. We continue to see marked improvement in our safety and environmental performance and we will achieve and maintain HS&E third party certification at the business and operational levels of the organization wherever this commitment has been made.

As responsible corporate citizens, we continue to renew our commitment to the public through outreach activities, and by proactively communicating with our surrounding communities.

[Signature of Andreas Kramvis]

Performance Materials and Technologies
President and CEO

[Signature of John A. Carter]

Performance Materials and Technologies
Vice President
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WARNING: BURNS WITH CONCENTRATED HF ARE USUALLY VERY SERIOUS, WITH THE POTENTIAL FOR SIGNIFICANT COMPLICATIONS DUE TO FLUORIDE TOXICITY. CONCENTRATED HF, LIQUID OR VAPOR MAY CAUSE SEVERE BURNS, METABOLIC IMBALANCES, PULMONARY EDEMA AND LIFE THREATENING CARDIAC ARRHYTHMIAS. EVEN MODERATE EXPOSURES TO CONCENTRATED HF MAY RAPIDLY PROGRESS TO FATALITY IF LEFT UNTREATED.

Introduction

Because the medical treatment of hydrofluoric acid exposure is so specialized and differs from the treatment of other inorganic acid exposures, physicians may be unaware of appropriate treatment measures. It is recommended that HF users ensure ahead of time that their local medical resources are familiar with the toxicity of HF and the treatment of HF exposure. This would include, at a minimum, thoroughly reviewing this booklet and making sure that treatment facilities and supplies are available.

Hydrofluoric acid (CAS # 7664-39-3) is very aggressive physiologically because of the fluoride ion. Both anhydrous hydrofluoric acid (hydrogen fluoride) and its solutions are clear, colorless liquids. When exposed to air, concentrated solutions and anhydrous hydrofluoric acid produce pungent fumes which are especially dangerous. Unless heated, dilute concentrations of hydrofluoric acid in water (e.g., less than 40% HF) do not produce significant vapor concentrations.

NOTE: Persons unfamiliar with *hydrofluoric acid* often mistake it for, or confuse it with, *hydrochloric acid*. Although hydrofluoric acid (HF) and hydrochloric acid (HCl) have similar sounding names, the toxicity of these two acids is very different. To decrease or avoid confusion, we recommend that HYDROFLUORIC ACID and HYDROGEN FLUORIDE be referred to as "HF".

HF is primarily an industrial raw material. It is used in fluorocarbon production, stainless steel manufacturing, metal finishing, aluminum manufacturing, inorganic and organic chemical manufacturing, petroleum refining, mineral processing, glassmaking and electronic components manufacturing. It is also used in certain industrial and consumer cleaning compounds. **However, its use in consumer products is discouraged because of its potential toxicity.**

Most non-industrial burns are caused by dilute concentrations of HF (e.g, less than 15% HF). Most of the HF used in the electronics industry is less than 50%. However, many industrial uses of HF involve concentrated (50-100%) HF.

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The recommended medical procedures described in this brochure are based on a review of the available literature, shared experiences with others who have dealt with the health effects of HF, the personal knowledge and experiences of Honeywell physicians, nurses and other professionals in dealing with the unique hazards of this product, and experimental laboratory work sponsored by Honeywell.

Every effort must be made to prevent exposure to HF. If exposure does occur, the specialized procedures which follow are recommended to avoid the very serious consequences that might otherwise occur.

Acute Toxicity

SKIN CONTACT

HF can cause serious, painful burns of the skin. Specialized first aid and medical treatment is required. **Burns larger than 25 square inches (160 square cm) may result in serious systemic toxicity.**

HF is a highly corrosive acid which can severely burn skin, eyes, and mucous membranes. The vapors from anhydrous hydrofluoric acid or its concentrated solutions can also burn these tissues.

HF is similar to other acids in that the initial extent of a burn depends on the concentration, the temperature, the duration of contact with the acid and the size of the burn. **Hydrofluoric acid differs, however, from other acids because the fluoride ion readily penetrates the skin, causing destruction of deep tissue layers. Unlike other acids which are rapidly neutralized, this process may continue for days if left untreated.**

Strong acid concentrations (over 50%), and particularly anhydrous HF (AHF or 100% HF), cause immediate, severe, burning pain and a whitish discoloration of the skin often followed by blister formation. Skin exposure to HF vapors can result in similar burns.

HF skin burns are usually accompanied by severe, throbbing pain which is thought to be due to irritation of nerve endings by increased levels of potassium ions entering the extracellular space to compensate for the reduced levels of calcium ions, which have been bound to the fluoride. **Relief of pain is an important guide to the success of treatment.**

The usual initial signs of an HF burn are redness, edema, and blistering. With more concentrated acids, a blanched white

area appears. The fluoride ion penetrates the upper layers of the skin. A thick granular exudate may form under blisters due to liquefaction necrosis. In rare (and untreated) cases, there may be penetration to underlying bone with decalcification.

HF burns require immediate and specialized first aid and medical treatment (2, 3, 4, 5, 6, 7) differing from the treatment of other chemical burns. If untreated or if improperly treated, permanent damage, disability or death may result. (8) If, however, the burns are promptly and properly recognized and managed, the results of treatment are generally favorable.

Treatment is directed toward binding the fluoride ions to prevent tissue destruction. High molecular weight quaternary ammonium compounds, e.g. benzalkonium chloride (Zephiran®), are used as soaking agents. (9, 10, 11) Calcium gluconate as a gel or ointment can be applied locally, and calcium gluconate solution may be injected (subcutaneously, intravenously, or intra-arterially), inhaled, or used as an irrigant. (3, 12, 13, 14, 15)

Speed is of the essence. Delays in first aid care or medical treatment or improper medical treatment will likely result in greater damage or may, in some cases, result in a fatal outcome. **During transportation to a medical facility or while waiting for a physician to see the victim, it is extremely important to continue the benzalkonium chloride (Zephiran) soaks or compresses or continue massaging calcium gluconate gel.** In contrast to the immediate effects of concentrated HF, the effects of contact with more dilute hydrofluoric acid or its vapors may be delayed, and this is one of the problems with the recognition of some HF burns. Skin contact with acid concentrations in the 20% to 50% range may not produce clinical signs or symptoms for one to eight hours. With concentrations less than 20%, the latent period may be up to twenty-four hours. HF concentrations as low as 2% may cause symptoms if the skin contact time is long enough. (1)

SYSTEMIC TOXICITY

To produce HF, calcium fluoride is reacted with sulfuric acid:



This production process requires a great deal of energy to accomplish. On the other hand, in the body:



This process releases energy, and therefore occurs very readily. The toxic effect of HF on body calcium is certainly more complicated than this. There is some evidence that fluoride may combine with calcium and phosphate, so that five calcium ions are tied up for each fluoride ion (e.g. $\text{Ca}_5\text{F}(\text{PO}_4)_3$), rather than two. There is also some evidence that there may be high intracellular levels of calcium in some tissues, rather than low levels, as would intuitively be expected. (16) However, the reaction of fluoride with body calcium is one of the major toxic effects and forms the basis for many treatment recommendations.

One of the most serious consequences of severe exposure to HF by any route is the marked lowering of serum calcium (hypocalcemia) and other metabolic changes, which may result in a fatal outcome if not recognized and treated. Hypocalcemia should be considered a potential risk in all instances of inhalation or ingestion, and **whenever skin burns exceed 25 square inches**, (160 square centimeters). Serum magnesium may also be lowered, and elevations in serum potassium have been reported to further complicate the metabolic imbalances which need to be monitored and corrected. (16, 17, 18) High levels of fluorides have been noted both in the blood and body organs. Hemodialysis has been reported to be effective therapy for cases of severe systemic fluoride intoxication. (19, 20, 21) Treatment for shock may also be required as for other severe injuries.

Other effects reported from fluoride exposure include coagulation defects and inhibition of a number of enzymes, including preglycolytic enzymes, phosphatases and cholinesterase. The results of this enzyme inhibition include inhibition of cellular glucose phosphorylation and subsequent glycolysis, inhibition of respiration, and increased sensitivity of cholinergic mechanisms to acetyl cholinesterase. (22)

While hypocalcemia has been traditionally considered the major systemic effect of severe poisoning with HF, it is apparent that hypomagnesemia, hyperkalemia, the cardiodepressing and vasodilating effects of fluoride and effects on pulmonary hemodynamics and systemic capacitance vessels, including an increase in pulmonary vascular resistance, all play a role in systemic toxicity. Although some of these effects have been described, the implications for therapeutic measures have not been well defined. (23, 24)

EYE CONTACT

Hydrofluoric acid can cause severe eye burns with destruction or opacification of the cornea. Blindness may result from

severe or untreated exposures. Immediate first aid and specialized medical care is required. (3, 13)

INHALATION

Hydrofluoric acid fumes may cause laryngospasm, laryngeal edema, bronchospasm and/or acute or delayed pulmonary edema. Acute symptoms may include coughing, choking, chest tightness, chills, fever and cyanosis. Many reported fatalities from HF exposures have been due to severe pulmonary edema (coupled with systemic toxicity) that did not respond to usual medical treatment.

Burns from vapors or liquid contact to the oropharyngeal mucosa or upper airway may cause severe swelling to the point of requiring a tracheostomy. It is recommended that all patients with such exposures be hospitalized for observation and/or treatment.

Because of the strong irritant nature of HF, an individual inhaling HF vapors or fumes will usually experience upper respiratory injury, with mucous membrane irritation and inflammation as well as cough. All individuals suspected of having inhaled HF should be observed for pulmonary effects. This would include those individuals with significant upper respiratory irritation, bronchoconstriction by pulmonary auscultation or spirometry, and any individual with HF exposure to the head, chest or neck areas. It has been reported that pulmonary edema may be delayed for several hours and even up to two days. If there is no initial upper respiratory irritation, significant inhalation exposure can generally be ruled out.

The Permissible Exposure Limit (PEL) set by the U.S. Occupational Safety and Health Administration (OSHA) is a time weighted average exposure for 8 hours of 3 ppm. (25) The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a ceiling level of 2 ppm or 1.53 mg/m³ with a 0.5 ppm TLV-TWA. (26) The National Institute for Occupational Safety and Health (NIOSH) has established the level that is immediately dangerous to life and health (IDLH) at 30 ppm. (27, 28) The American Industrial Hygiene Association has published an Emergency Response Planning Guideline setting 50 ppm as the maximum level below which nearly all individuals could be exposed for one hour without experiencing or developing life-threatening health effects (ERPG-3), 20 ppm as the maximum level below which nearly all individuals could be exposed for one hour without developing irreversible health effects or symptoms which would impair taking protective action (ERPG-2), and 2 ppm as the maximum level below

which nearly all individuals could be exposed up to one hour without experiencing other than mild, transient adverse health effects (ERPG-1). (29)

The California Occupational Safety and Health Standards Board has established a PEL of 0.4 ppm 8hr TWA [0.33mg/m³] with a STEL (Short term exposure level) of 1 ppm [0.83mg/m³]. (32)

INGESTION

Ingestion of HF may result in severe burns to the mouth, esophagus and stomach. Severe systemic effects are common. Ingestion of even small amounts of dilute HF have resulted in death. (30)

Toxic myocarditis, believed to be associated with HF Acid ingestion with a suicide attempt has been reported. (5)

Chronic Toxicity

Because it is a strong irritant there are few exposures to HF sufficient to suggest risk of chronic toxicity. HF has not been the subject of long term toxicity studies or testing. Once HF enters the body, it is expected that the fluoride ion would be the major concern from a chronic toxicity standpoint. Chronic toxicity from long term, high exposure to fluoride salts (eg. SnF₂, NaF, Na₂FPO₃) has been reported to result in tooth mottling in children, bone fluorosis and sometimes osteosclerosis in adults and children.

Skeletal fluorosis is known to be associated with excessive exposure to fluoride compounds. Cases of skeletal fluorosis have been reported in populations exposed to naturally occurring drinking water containing greater than 10 ppm of fluoride ion and in individuals exposed to high levels of fluoride containing dusts. However, skeletal fluorosis has not been reported as a consequence of HF exposure.

Because of the use of fluoride to prevent dental caries, there is ongoing evaluation of fluorides for the potential to cause cancer. There is no evidence that fluoride is genotoxic except in some *in vitro* assays at cytotoxic concentrations. Epidemiological studies have not demonstrated an association between fluoride in drinking water and an increase in cancer. The International Agency for Research on Cancer (IARC) has not classified hydrogen fluoride as to its human carcinogenicity, and neither fluorides nor HF are listed by IARC, NTP, OSHA, ACGIH, NIOSH, the State of California or

other governmental agencies as causing cancer. (31, 32, 33) In animal studies, fluoride salts have caused effects in off-spring only at high, maternally toxic levels. Some animal studies have shown effects on male fertility, e.g. decreased sperm counts. (33) Fluoride exposures should be kept below recommended levels to assure no adverse effects to the developing fetal skeletal system or teeth.

Monitoring of urine for fluorides is an accepted method of determining exposure. (34) Urine fluoride levels above 3 mg/liter at the beginning of a workshift, or above 10 mg/liter at the end of a workshift, may indicate excessive absorption of fluoride. It should be noted that fluorides are often present in significant amounts in persons not occupationally exposed (because of dietary sources of fluoride such as tea), and that the urine fluoride determination is not specific for HF. (26)

First Aid Treatment for Hydrofluoric Acid Burns

In Case of Contact or Suspected Contact with HF:

SKIN CONTACT

1. Move victim immediately under safety shower or other water source and flush affected area thoroughly with large amounts of running water. Speed and thoroughness in washing off the acid is of primary importance.
2. Begin flushing even before removing clothing. Remove all contaminated clothing while continuing to flush with water.
3. Rinse with large amounts of running water. If 0.13% benzalkonium chloride (Zephiran) solution or 2.5% calcium gluconate gel are available, the rinsing may be limited to 5 minutes, with the soaks or gel applied as soon as the rinsing is stopped. If benzalkonium chloride (Zephiran) or calcium gluconate gel is not available, rinsing must continue until medical treatment is rendered.
4. While the victim is being rinsed with water, someone should alert first aid or medical personnel and arrange for subsequent treatment.
5. Immediately after thorough washing, use one of the measures below:
 - a. Begin soaking the affected areas in iced 0.13% benzalkonium chloride (Zephiran) solution.

Use ice cubes, not shaved ice, in order to prevent frostbite.

If immersion is not practical, towels should be soaked with iced 0.13% benzalkonium chloride (Zephiran) solution and used as compresses for the burned area. Compresses should be changed every two to four minutes. Do not use benzalkonium chloride (Zephiran) solution for burns of the eyes. Exercise caution when using benzalkonium chloride (Zephiran) solution near the eyes as it is an eye irritant. Benzalkonium chloride (Zephiran) soaks or compresses should be continued until pain is relieved or until more definitive medical treatment is provided.

- b. Start massaging 2.5% calcium gluconate gel into the burn site.

Apply gel frequently and massage continuously until pain and/or redness disappear or until more definitive medical care is given.

The individual applying the calcium gluconate gel should wear surgical gloves to prevent a possible secondary HF burn.

NOTE: Clinical experience has shown that both benzalkonium chloride (Zephiran) and calcium gluconate gel are effective when used correctly in appropriate situations. In an animal model, benzalkonium chloride (Zephiran) soaks are superior to calcium gluconate gel under the experimental conditions used. (37, 38)

6. After treatment of burned areas is begun, the victim should be examined to ensure there are no other burn sites which have been overlooked.
7. Arrange to have the victim seen by a physician. (If burns are small and/or caused by weak acid, and treatment has been provided by an experienced individual, evaluation by a physician may not be necessary.) **During transportation to a medical facility or while waiting for a physician to see the victim, it is extremely important to continue the benzalkonium chloride (Zephiran) soaks or compresses or continue massaging calcium gluconate gel.** In many situations, particularly for minor burns covering a small skin area or for burns caused by dilute HF, continued treatment with soaks or gel may be effective as the sole type of medical care. All persons with extensive burns or burns with significant blister formation or with the appearance of whitish or dead

skin need to be seen by a physician. All persons with HF burns which do not respond to either calcium gluconate gel or benzalkonium chloride (Zephiran) soaks or compresses within 30 minutes should be evaluated by a physician.

8. The physician may advise continuation of benzalkonium chloride (Zephiran) soaks or calcium gluconate gel.
 - a. If the physician advises continued treatment with benzalkonium chloride (Zephiran) soaks or compresses, the soaks or compresses are usually required for 2 to 4 hours. Significant relief of pain should be noted within the first 30 minutes. If this does not occur, the victim must be seen by a physician and more definitive care instituted. If the pain is substantially relieved within the first 30 minutes, continue the treatment for a total of 2 hours. After that time, discontinue treatment and observe for the recurrence of pain. If pain recurs, continue soaks or compresses until relief of pain occurs. Soaking for 6 hours is sometimes needed. (Note: Because prolonged immersion in the ice bath may result in discomfort, relief may be obtained by removing the part from the bath every 10 minutes for a minute or so and then reimmersing it. After the initial 30-60 minutes of treatment, less ice can be used so the bath is cool rather than cold.)
 - b. *Calcium gluconate gel* may be used for several hours or even repeated over a period of a few days. However, if significant relief of pain does not occur within 30 to 40 minutes, more definitive treatment such as Calcium Gluconate injections or iced benzalkonium chloride (Zephiran) will be required. For small burns, or burns of the face, ears, and near mucous membranes, calcium gluconate gel may be very useful. The gel is applied frequently and massaged into the burned area. This is continued until relief is obtained or further medical care is available.

9. For serious burns, medical attention must be provided as quickly as possible.

For minor burns, if first aid treatment does not alleviate symptoms or if symptoms persist or recur, medical attention must be sought.

EYE CONTACT

1. **Immediately** flush the eyes for at least 15 minutes with large amounts of gently flowing water. Hold the eyelids open and away from the eye during irrigation to allow thorough flushing of the eyes. **Do not use the benzalkonium chloride (Zephiran) solutions described for skin**

treatment. If the person is wearing contact lenses, the lenses should be removed, if possible. However, flushing with water should not be interrupted, and the lenses should be removed by a person who is qualified to do so. If sterile 1% calcium gluconate solution is available, **water washing may be limited to 5 minutes**, after which the 1% calcium gluconate solution should be used to irrigate the eye using a syringe or a continuous irrigation device.

2. Take the victim to a doctor, preferably an eye specialist, as soon as possible. Ice water compresses may be applied to the eyes while transporting the victim to the doctor.
3. If a physician is not immediately available, apply one or two drops of 0.5% tetracaine hydrochloride, 0.5% proparacaine, or other aqueous, topical ophthalmic anesthetic and continue irrigation. Use no other medications unless instructed to do so by a physician. Rubbing of the eyes is to be avoided.

INHALATION

1. Immediately move victim to fresh air and get medical attention.
2. Keep victim warm, quiet and comfortable.
3. If breathing has stopped, start artificial respiration at once.
4. 100% Oxygen should be administered as soon as possible by a trained individual. Continue oxygen while awaiting medical attention unless instructed otherwise by a physician.
5. A nebulized solution of 2.5% calcium gluconate may be administered with oxygen by inhalation.
6. Do not give stimulants unless instructed to do so by a physician.
7. The victim should be examined by a physician and held under observation for at least a 24 hour period.

INGESTION

1. Have the victim drink several large glasses of water or milk to dilute the acid. **Do not induce vomiting.** Do not give emetics or baking soda. Never give anything by mouth to an unconscious person.
2. Give several glasses of milk or several ounces of milk of magnesia, any calcium containing antacid®, Maalox®, etc or grind up and administer up to 30 Tums™, Caltrate® or other antacid tablets with water. The calcium or magnesium in these compounds may act as an antidote, however this has not been supported in the literature (39).

3. Get immediate medical attention. Ingestion of HF is a life-threatening emergency.

Medical Treatment for Hydrofluoric Acid Burns

BURNS OF THE SKIN – GENERAL

Burns from dilute acid are difficult to distinguish from other chemical burns and usually appear as areas of erythema. However, they may progress, if not treated, to areas of blistering, necrosis or ulceration. Burns from more concentrated acid have a characteristic appearance and present as severely reddened, swollen areas with blanched, whitish regions which rapidly progress to blistering and necrosis. A thick granular exudates usually appears under these blisters and requires debridement and removal.

Concentrated HF burns cause extreme pain. The pain is thought to result from nerve ending irritation due to increased levels of potassium ions in extracellular spaces to compensate for the reduced levels of calcium ions which have been bound by the fluoride. **Relief of pain is an excellent indication of the success of treatment and, therefore, local anesthetics should be avoided.**

Many different types of therapies have been suggested for HF burns. The aim of all treatment is to chemically sequester the fluoride ion and to prevent extensive, deep-tissue destruction. (37, 38)

After treatment of recognized burned areas is begun, the victim should be carefully examined to insure there are no other burn sites which may have been overlooked.

QUATERNARY AMMONIUM COMPOUNDS

Most HF burns can be satisfactorily treated by immersion of the burned part in an iced, aqueous solution of a quaternary ammonium compound. Two solutions have been clinically successful, 0.13% benzalkonium chloride (e.g. Zephiran) or 0.2% benzethonium chloride. Because of its availability as a non-prescription drug, benzalkonium chloride (Zephiran) is recommended in the United States.

The solutions should be cooled with ice cubes. (Shaved or crushed ice may cause excessive cooling, with the danger of frostbite.)

If immersion in the solution is not practical, soaked compresses of the same iced solution should be applied to the burned area. The immersion or compresses should be used for **at least two hours**. Compresses should be changed or soaked with additional solution approximately every two to four minutes.

If blisters are present, they should be opened and drained and necrotic tissue should be debrided by a physician or qualified health care practitioner as soon as possible. However, immersion in benzalkonium chloride (Zephiran) or use of compresses should not be delayed if debridement cannot be accomplished immediately.

Prolonged immersion in the iced benzalkonium chloride (Zephiran) bath may result in discomfort due to excess chilling; relief may be obtained by removing the burned part from the bath every ten to fifteen minutes for a few minutes and then reimmersing it when pain recurs. After the initial 30-60 minutes of treatment, less ice can be used so the bath is cool rather than cold.

The success of this treatment is indicated by relief of the severe pain in the burned area. If there is no significant relief of pain within 30 to 40 minutes, the use of 2.5 - 5% calcium gluconate injections may be necessary. If pain recurs when the treatment is stopped at the end of the first two hours, immersion or compresses should be resumed until pain is relieved. A total of four to six hours immersion or use of compresses of benzalkonium chloride (Zephiran) may be required for the treatment of most burns. No further iced Zephiran treatment will be required in many instances. The use of iced quaternary ammonium compound solutions offers several advantages over topical calcium gluconate gel:

- ability to treat burns on multiple surfaces, such as the hand, more efficiently;
- reduction of local pain;
- possible slowing of the rate of tissue destruction;
- possible slowing of the passage of the fluoride ion into deeper tissues and into the bloodstream;
- does not require continuous massaging.

Large burns, serious burns due to concentrated HF, or burns with delayed treatment will probably require the use of calcium gluconate injections in addition to or instead of the benzalkonium chloride (Zephiran) soaks.

Quaternary ammonium compounds should not be used for burns on the face, ears or other sensitive areas due to their irritating nature. It is preferable to use calcium gluconate gel or calcium gluconate injection in these areas.

CALCIUM GLUCONATE GEL

Calcium gluconate gel, consisting of 2.5% USP calcium gluconate in a surgical water soluble lubricant, is widely used for first aid and/or primary treatment of HF burns of the skin. The gel is convenient to carry and can be used to initially treat small burns that might occur away from medical care. (The gel is not recommended for burns with concentrated HF except as a first aid measure.) The gel is used by massaging it promptly and repeatedly into the burned area, until pain is relieved. If possible, surgical gloves should be worn during initial application of the gel, so the person providing treatment will not receive a secondary HF burn. This treatment can be started without waiting for medical direction. Several commercially available calcium gluconate gel formulations have been evaluated and found to give comparable outcomes. (35)

If used as the only method of treatment, liberal quantities of calcium gluconate gel must be massaged into the burned area continuously for up to several hours. Relief of pain can be used to assess the efficacy of this treatment. If good relief of pain is not obtained after 30-40 minutes, alternate methods of treatment such as calcium gluconate injections or benzalkonium chloride (Zephiran) soaks should be considered.

The gel is especially useful for burns on the face, particularly near the mouth, eyes, on the ears, or for small, dilute acid burns elsewhere. It may be convenient to use the gel for very small burns where the victim can easily apply and massage the gel into the burned area. Use of the gel may be more convenient for dilute acid burns such as occur with commercial products like rust removers, aluminum cleaners or etching solutions.

CALCIUM GLUCONATE INJECTIONS

After first aid measures have been taken, injection of a 2.5% - 5% calcium gluconate solution is indicated as the primary medical treatment for large burns (over 25 square inches or 160 square centimeters). For smaller burns, if benzalkonium chloride (Zephiran) soaks or calcium gluconate gel do not result in significant relief of pain within 30 to 40 minutes, injection of calcium gluconate solution is indicated. Injection of calcium gluconate solution may also be indicated for burns in which treatment has been delayed.

The physician should inject sterile 2.5 - 5% aqueous calcium gluconate beneath, around and into the burned area. Calcium gluconate is packaged as a 10% solution, and must be diluted 50:50 or 25:75 with normal saline to make 5% or 2.5% solutions. (Note: DO NOT USE calcium chloride, which is corrosive and may result in additional damage.)

If subcutaneous calcium gluconate injections are used, the amount injected initially is small and should not exceed 0.5 cc per square centimeter of affected skin surface. The injections should not distort the appearance of the skin. A small-gauge needle (27-30 gauge) should be used, and the burned area should be injected through multiple sites. With successful treatment, pain relief following injection of 2.5% - 5% calcium gluconate solution is very rapid. The patient can usually advise when the pain stops, and this is an indicator of adequate treatment. Multiple injections in skin that has compromised integrity may increase the risk of infection, and the use of antibiotic creams such as Silvadene® (silver sulfadiazine) or Garamycin® (gentamicin sulfate cream) should be considered following such treatment. Local anesthetics should not be used since they mask pain relief which is an important indication of adequacy of treatment.

Some physicians prefer using calcium gluconate injections initially as the primary treatment, instead of using quaternary ammonium compound soaks or compresses or calcium gluconate gel. Injections often are not necessary when there has been early and adequate treatment with soaks or gel.

CALCIUM GLUCONATE SOLUTION

In some instances, a 5% or 10% calcium gluconate solution may be used in compresses or for irrigation. For example, irrigating with a calcium gluconate solution may be the best treatment should HF enter the external ear canal. In this instance, referral to an otolaryngologist may also be needed.

BURNS OF THE FINGERS AND NAILS

Burns of the fingers often create special problems in treatment. Finger and toe nails permit penetration of fluoride ions but prevent soaks or gels from being effective. It may be necessary to drill, split or even remove nails to allow the topical methods of treatment to be effective. One author has cautioned that removal of the nail should rarely be necessary in the case of dilute HF acid (less than 10%) burns. (40) The treating physician must consider the

morbidity associated with removal of the nail versus the need to treat the HF exposure.

If immersion in benzalkonium chloride (Zephiran) solution is started immediately, it may be possible to avoid removing the nail. Sometimes better penetration under the nail can be successfully accomplished by splitting the nail or by drilling several burr holes in the nail using a large gauge needle or a nail drill. If calcium gluconate injection is used as treatment, the nail may still need to be split or removed. If nail removal is necessary, using a short acting regional or ring-block anesthetic may facilitate this procedure and not interfere with using pain relief as an indicator of effective treatment. When using calcium gluconate injections in the digits, care must be taken to inject the solution cautiously so as to avoid compromising the circulation in these areas.

If benzalkonium chloride (Zephiran) soaks are not available, experience has shown that some finger or hand burns can be treated by using a glove filled with calcium gluconate gel. Initially, calcium gluconate gel should be massaged into the burned area. Following this, an oversize surgical glove should be partially filled with calcium gluconate gel, and the hand inserted into the glove. The gloved hand may be immersed in ice water, if available, which may aid pain relief. This treatment works best for burns where there is no blistering, or after the burns have been debrided. As in other cases where calcium gluconate gel is used, alternate methods of treatment should be considered if good relief of pain is not achieved within 30 - 45 minutes. If pain is relieved, the glove should remain in place for three to four hours.

INTRA-ARTERIAL AND INTRAVENOUS CALCIUM INFUSION

Reports in the literature have described the use of intra-arterial injection or infusion of dilute calcium gluconate solutions to treat HF burns of the hand and digits. This method, although rather involved, should be considered in selected cases, especially where inadequate or delayed treatment has occurred. The method is described as follows:

“A long catheter was inserted percutaneously into the radial artery using standard aseptic technique. Intra-arterial catheter placement was confirmed by pressure transducer and oscilloscope. If the burn involved only the thumb, index, or long fingers, the catheter was advanced only a few centimeters proximally in preparation for digital subtraction arteriography. If the burn involved the ring or small fingers, the catheter was advanced proximally into the brachial artery because access to the ulnar circulation was necessary.

Following satisfactory placement of the arterial catheter, digital subtraction arteriography was performed on all patients in our series to identify the origin of vascular supply to digits involved.

Once the tip of the arterial catheter was in the desired location, a dilute preparation of calcium gluconate (10 ml of a 10% solution mixed in 40 to 50 ml 5% dextrose) was infused with a pump apparatus into the catheter over four hours. Each patient was observed closely during the infusion period for progression of symptoms and potential complications of the procedure, such as alterations of distal vascular supply.

Following the four-hour infusion, the arterial catheter was maintained in place in the usual manner while the patient underwent an observation period. If typical HF pain returned within four hours, a second calcium infusion was repeated until the patient was pain free four hours following completion of the calcium infusion”. (14)

There are now several reports of the successful use of intravenous calcium gluconate to treat HF burns of the upper extremity. (41, 42, 43) Graudins, et al. describe their method:

An intravenous catheter was placed on the dorsum of the affected hand. The superficial veins were exsanguinated by elevation. A double-cuffed pneumatic tourniquet was applied above the elbow, inflated to 100 mm Hg above systolic blood pressure, and 10 ml of 10% calcium gluconate diluted with 30 to 40 ml of 0.9% saline solution was then infused. Ischemia was maintained for 25 minutes; the cuff was sequentially released over 3 to 5 minutes.

This Bier Block method was most successful for burns due to dilute acid. If the use of intravenous calcium gluconate was not successful in relieving pain (which occurred with burns due to 49% HF, the highest concentration seen in the series of patients), Graudins et al. turned to intra-arterial calcium gluconate infusion.

ADDITIONAL MEASURES

HF burns are very aggressive and it is extremely important that there be no delays during decontamination, first aid or medical treatment. Assuring continuous care after decontamination is critical to ensure a good result from medical treatment.

In instances of extensive burns, skin grafting has occasionally been required, but the need for this treatment should be markedly reduced by immediate and aggressive primary treatment.

Follow-up care requires monitoring to prevent secondary infections. The use of daily dressings with antibiotic creams such as Silvadene or Garamycin has proven effective. HF burns may heal slowly, but if properly treated most heal with little or no scarring in 14 to 28 days.

ADDITIONAL AND UNPROVEN THERAPIES

The use of intravenous calcium gluconate is discussed above. Both Williams, *et al.* (44) and Cox, *et al.* (45) have discussed the use of intravenous magnesium sulfate to treat localized moderate to serious skin burns. Using either a rat or a rabbit model, the authors administered intravenous magnesium sulfate. Cox used a 0.2 mEq bolus over two minutes, followed by a slower infusion of 0.2 mEq per hour for four hours, with a total of 1.0 mEq/kg magnesium sulfate administered. Williams administered 8 mg/kg over five minutes or 160 mg/kg over 10 minutes. These authors compare this dose to the amount of magnesium sulfate, infused more slowly, used in the treatment of eclampsia.

Dunn, *et al.* (37) have shown effectiveness of locally applied calcium acetate solution, 10% in water at room temperature, in an animal model.

Seyb *et al.* (47), performed an experiment in rats using a topically applied solution of 50% aqueous dimethyl sulfoxide (DMSO) containing calcium gluconate (20% wt/vol). This treatment gave results comparable to injecting 10% calcium gluconate or 10% magnesium sulfate, and was superior to calcium gluconate gel in treating experimental HF burns.

It should be noted that many of these therapies, while promising, have been tested to a limited degree, if at all, in humans. A product developed in France, "Hexafluorine" (46), has been marketed in Europe and the United States for use as a decontamination solution for HF skin and eye exposure. Honeywell has conducted animal studies on this product with equivocal test results. Scientific documentation of effectiveness and experience with this product are lacking at this time and we therefore do not recommend its use (48).

SYSTEMIC ABSORPTION AND METABOLIC EFFECTS

Significant amounts of fluoride ion may be absorbed by skin contact, inhalation, or by ingestion. If systemic absorption of fluoride occurs, hypocalcemia, hypomagnesemia and hyperkalemia may also occur. All of these parameters need to be monitored and appropriate therapeutic measures instituted. The patient should be observed for clinical signs of hypocalcemia following ingestion or inhalation or following extensive burns greater than 25 square inches. **Serum calcium determinations must be performed** immediately and periodically to monitor and treat hypocalcemia. Severe lowering of serum calcium levels can occur within one to two hours even with HF burns covering less than 2.5% of body surface area. (8) Continuous EKG monitoring to observe prolongation of the Q-T interval may be useful to detect early changes in serum calcium, although profound hypocalcemia following HF exposure has been reported in the absence of EKG changes or in the absence of other signs of tetany.

The fall in serum calcium may occur precipitously following HF exposure. In two reported cases of exposure to anhydrous HF, the serum calcium fell to levels around 3 milliequivalents per liter (mEq/L) [normal = 8.8 - 10.3 mEq/L] within one to three hours of exposure. (8)

If necessary, aqueous calcium gluconate may be given intravenously. Calcium gluconate as a 10% solution must be given slowly since excess calcium can produce vagal bradycardia, ventricular arrhythmias and ventricular fibrillation. The IV calcium gluconate should be repeated until serum calcium levels return to, and remain at, normal levels. In one fatal case, 280 mEq of calcium over four hours was not sufficient to correct the profound hypocalcemia. (8) Without additional measures such as hemodialysis, it may not be possible to correct extreme hypocalcemia.

Serum magnesium levels should also be monitored and magnesium loss should be replaced intravenously if indicated. Yamaura, *et al.* have reported a case of HF exposure in which prolonged QT interval occurred, in which ionized calcium levels were relatively high but the magnesium level was low. (49) Serum potassium must also be carefully monitored. Significant elevations of serum potassium have been noted in cases of fluoride toxicity and also in laboratory studies. Hyperkalemia has also been implicated as a causative factor in cardiovascular collapse, and should be treated appropriately.

Even with normalization of serum calcium and potassium, life threatening ventricle arrhythmia may occur, possibly due to a direct toxic effect of the fluoride ion on the myocardium. (36).

HEMODIALYSIS with fluoride free water (and normal to low potassium and slightly higher calcium concentrations), in conjunction with other treatments mentioned, should be considered in all cases of serious burns and may need to be repeated if indicated. (19, 20, 21) Serum fluoride levels should be monitored. Normal plasma fluoride levels may differ because of various methodologies and analytical techniques. The decision to use dialysis should be based on the HF exposure (concentration, body surface area) and the clinical condition of the patient, including the serum levels of fluoride, calcium and potassium.

Primary excision or surgical removal of tissue has been recommended by some practitioners as a method of reducing systemic absorption of fluoride. (50) While this could in some instances be life saving, it is a rather drastic measure. It is likely that renal dialysis could be used to effectively treat systemic toxicity and would not result in the disfigurement, disability, or morbidity which could be associated with primary excision.

EYE INJURIES

HF can cause severe eye burns, which, if not properly treated, may result in scarring and blindness. The prognosis is not good if first aid treatment is delayed or inadequate. After first aid treatment (see FIRST AID section) the following medical treatment may be provided:

If the individual wears contact lenses, it is usually best to remove the lenses before additional eye irrigation.

Mix 50 ml of 10% calcium gluconate with 500 ml of normal saline to give approximately a 1% calcium gluconate solution. After administering local anesthetic eye drops, use an eye clamp and IV infusion set or a two pronged nasal oxygen cannula to instill the solution over a period of one to two hours. Other irrigation devices, such as a Morgan Lens may be utilized as well. More prolonged use of the solution could possibly damage the cornea. Consult an ophthalmologist regarding additional treatment.

INHALATION INJURIES

Patients with inhalation exposures should also be observed for signs of systemic absorption and fluoride toxicity.

Exposure to hydrofluoric acid fumes can cause acute respiratory irritation, bronchospasm, and/or **pulmonary edema**. Medical personnel should also be alert to the possibility of development of pulmonary edema when extensive burns of the face, neck or chest have occurred. Intubation should be avoided, if possible.

The victim should be removed from exposure and administered 100% oxygen immediately. The use of 2.5% aqueous calcium gluconate given by nebulizer with 100% oxygen, or with intermittent positive pressure, has been recommended. Theoretically, this should reduce toxicity and damage from the fluoride ion and should be seriously considered in cases of inhalation exposure.

Repeated use of nebulized calcium gluconate, every 4 hours for 48 hours after a significant inhalation exposure, has been described. (51)

Burns of the oral mucosa or upper airway may cause severe swelling and necessitate a tracheostomy. It is, therefore, recommended that all such patients be admitted to a hospital for observation.

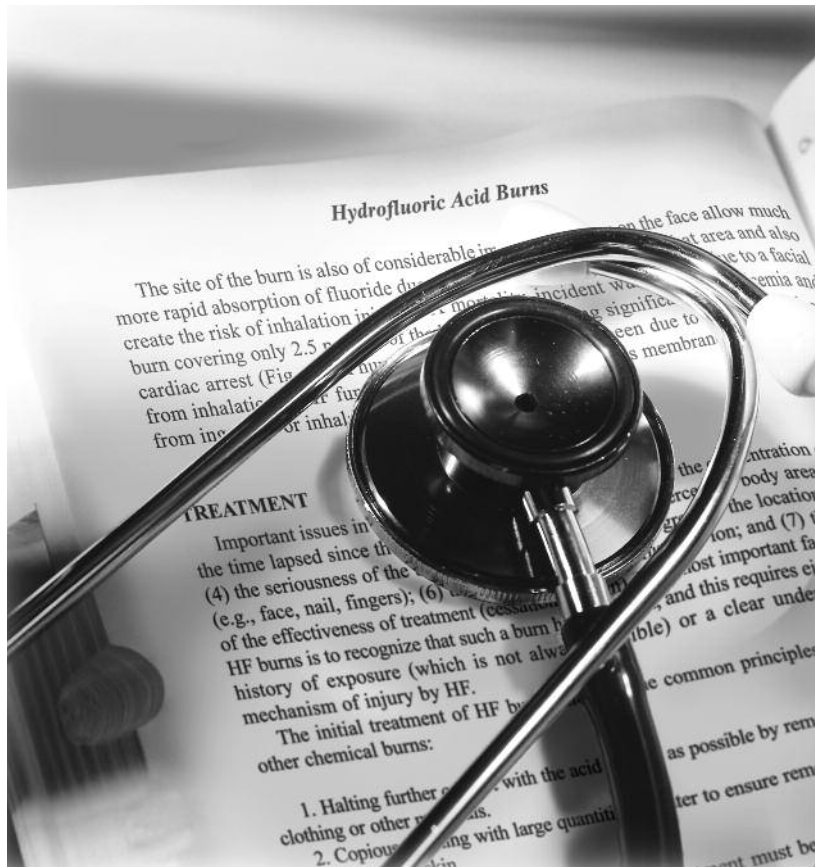
Because inhalation of HF may be associated with significant bronchospasm, inhaled, oral or parenteral bronchodilators should be administered as necessary. Even in the absence of symptoms, the prophylactic administration of inhalational steroids (e.g. beclomethasone dipropionate) may be indicated. (21) Pulmonary function testing may be helpful in assessing the degree and progress of pulmonary injury.

Specific measures may be needed to treat pulmonary edema.

INGESTION INJURIES

After first aid is completed (drinking several glasses of water followed by two glasses of milk or two ounces of milk of magnesia, or other calcium or magnesium containing antacids), the stomach may be lavaged with a solution of a calcium

containing antacid. The Lavage tube must be passed with care to prevent perforation. Treatment for the corrosive effects is the same as for ingestion of other strong acids. Systemic toxicity is very likely to occur and may require aggressive treatment.



References

1. Derelanko, M. J., et al.: Acute Dermal Toxicity of Dilute Hydrofluoric Acid. *J Toxicol-Cut and Ocular Toxicol* 4:73-85, 1985.
2. MacKinnon, M. A.: Hydrofluoric Acid Burns. *Dermatologic Clinics* 6:67-74, January, 1988.
3. Trevino, M. A.: Treatment of Severe Hydrofluoric Acid Exposures. *J Occup Med* 25:861-3, December, 1983.
4. Edelman, P.: Hydrofluoric Acid Burns. *State of the Art Rev Occup Med* 1:89-103, 1986.
5. Upfal, M. and Doyle, C.: Medical Management of Hydrofluoric Acid Exposure. *J Occup Med* 32: 726-731, August, 1990.
6. Caravati, E. M.: Acute Hydrofluoric Acid Exposure. *Am J Emerg Med* 6: 143-50, March, 1988.
7. ATSDR: Managing Hazardous Materials Incidents. *Medical Management Guidelines for Acute Chemical Exposure: Hydrogen Fluoride*. 2011.
8. Tepperman, P. B.: Fatality Due to Acute Systemic Fluoride Poisoning Following a Hydrofluoric Acid Skin Burn. *J Occup Med* 22:691-2, October, 1980.
9. Wetherhold, J. M.: Treatment of Hydrofluoric Acid Burns. *J Occup Med* 7:193-5, May, 1965.
10. MacKinnon, M. A.: Hydrofluoric Acid Burns. *Dermatologic Clinics* 6(1):67-74, 1988.
11. Reinhardt, C. F.: Hydrofluoric Acid Burn Treatment. *Am Ind Hyg Assoc J* 27:166-171, 1966.
12. Browne, T. D.: The Treatment of Hydrofluoric Acid Burns. *J Soc. Occup Med* 24:80-9, July, 1974.
13. Rose, L. and Trevino, M. A.: Further Evaluation of Hydrofluoric Acid Burns of the Eye. *J Occup Med* 26:483-4, July, 1984.
14. Vance, M. V.: Digital Hydrofluoric Acid Burns: Treatment with Intra-arterial Calcium Infusion. *Ann Emerg Med* 15:59-65, August 8, 1986.
15. Davanzo, F. et al.: Hydrofluoric Acid Intoxication: A New Therapy. *Med Lav* 78:333-6, 1987.
16. Boink, A. B. T. J.: Pathophysiological Mechanisms of Hydrofluoric Acid and Fluoride Intoxication: An Explorative Study in Rats and Pigs. Thesis. University of Utrecht, The Netherlands. 1993.
17. Mclvor, M. E.: Delayed Fatal Hyperkalemia in a Patient with Acute Fluoride Intoxication. *Ann Emerg Med* 16:1166-7, October, 1987.
18. Cummings, C. and Mclvor, M. E.: Fluoride Induced Hyperkalemia: The Role of Ca²⁺ Dependent K⁺ Channels. *Am J Emerg Med* 16:1-3, January, 1988.
19. Björnham, J. et al.: Hydrofluoric Acid - Induced Burns and Life Threatening Systemic Poisoning - Favorable Outcome After Hemodialysis. *J Toxicol Clin Toxicol* 41(6):855-60, 2003.
20. Mclvor, M. E.: Acute Fluoride Toxicity: Pathophysiology and Management. *Drug Saf* 5:79-85, 1990.
21. CTEF (Comité Technique Européen du Fluor, a sector group of CEFIC): *Medical Management of Hydrogen Fluoride Injuries*. February 2004.
22. Hazardous Substances Data Bank (HSDB): Sodium Fluoride., National Library of Medicine, 1992.
23. Gaugl, J.F. and Woolridge, B.: Cardiopulmonary Response to Sodium Fluoride Infusion in the Dog. *J Toxicol Environ Health* 11:765-82, 1983.
24. Strubelt, O., et al.: The Pathophysiological Profile of the Acute Cardiovascular Toxicity of Sodium Fluoride. *Toxicology* 24:313-23, 1982.
25. US Department of Labor (OSHA): CFR 1910.1000, Table Z-2.
26. 2011 Threshold Limit Values (TLVs®) for Chemical Substances and Physical Agents and Biological Exposure Indices (BEIs®), American Conference of Governmental Industrial Hygienists, Inc., Cincinnati, Ohio, 2011.
27. NIOSH Pocket Guide to Chemical Hazards. p 168 DHHS (NIOSH) Publication No. 97-140, June 1997.
28. Documentation for Immediately Dangerous to Life or Health Concentrations (IDLHs). pp 257-8 DHHS (NIOSH). NTIS Publication No. PB-94-195047, May 1994.
29. Emergency Response Planning Guideline: Hydrogen Fluoride. American Ind Hygiene Assn, 2008.
30. Cordero, S.C. et al.: A Fatality Due to Ingestion of Hydrofluoric acid. *J Anal Toxicol* 28 (3):211-13, 2004.
31. The Regulated Chemicals List of Lists (LOLI), ChemADVISOR®, Micromedex, Inc., 1998.
32. www.dir.ca.gov/oshsb/airborn_contaminants_2011.html
33. Chemical Substances Bureau, The Netherlands. Hydrogen Fluoride, Risk Assessment. Draft, 12 August, 1997 (with changes 24 November, 1997).
34. Kono, K., et al.: Urinary Fluoride Monitoring of Industrial Hydrofluoric Acid Exposure. *Environ Res* 42:415-20, April 1987.
35. Roblin I. et al.: Topical Treatment of Experimental Hydrofluoric Acid Skin Burns by 2.5% Calcium Gluconate. *J Burn Care Res* 2006; 27(6):889-94.
36. Vohra, R. et al.: Recurrent Life-Threatening Ventricular Dysrhythmia Associate with Acute Hydrofluoric Ingestion; Observations in One Case and Implications for Mechanism of Toxicity. *Clinical Toxicology* 2008;46:79-84.
37. Dunn, B. J., et al.: Hydrofluoric Acid Dermal Burns: An Assessment of Treatment Efficacy Using an Experimental Pig Model. *J Occup Med* 34:902-9, 1992.
38. Dunn, B. J., et al.: Topical Treatments for Hydrofluoric Acid (HF) Dermal Burns: Further Assessment of Efficacy Using an Experimental Pig Model. *J Occup Environ Med* 38: 507-14, 1996.
39. Heard, K. and Delgado, J.: Oral Decontamination with Calcium or Magnesium Salts Does Not Improve Survival Following Hydrofluoric Acid Ingestion. *J Toxicol Clin Toxicol* 41(6):789-92, 2003.
40. Roberts J. R. and Merigian, K. S.: Acute Hydrofluoric Acid Exposure (letter). *Am J Emerg Med* 7:125-6, January, 1988.
41. Henry, J. A. and Hla, K. K. Intravenous Regional CalciumGluconate Perfusion for Hydrofluoric Acid Burns. *Clin Toxicology* 30:203-7, 1992.
42. Gaudins, A., et al.: Regional Intravenous Infusion of Calcium Gluconate for Hydrofluoric Acid Burns of the Upper Extremity. *Ann Emerg Med* 30:604-7, 1997.
43. Ryan, J.M. et al.: Regional Intravenous Infusion of Calcium Gluconate for Hydrofluoric Acid Burns of the Upper Extremity. *Ann Emerg Med* 31:526-7, 1998.
44. Williams, J. M. et al.: Intravenous Magnesium in the Treatment of Hydrofluoric Acid Burns in Rats. *Ann Emerg Med* 23:464-9, 1994.
45. Cox, R. D. and Osgood, K. A. Evaluation of Intravenous Magnesium Sulfate for the Treatment of Hydrofluoric Acid Burns. *Clin Toxicology* 32:123-36, 1994.
46. Hexafluorine Product Literature, DH Marketing, Tarrytown, NY. 1993.
47. Seyb, S. T.: A Study to Determine the Efficacy of Treatments for Hydrofluoric Acid Burns. *J Burn Care Rehabil* 16:253-7, 1995.
48. Hulten, P. et al.: Hexafluorine vs Standard Decontamination to Reduce Systemic Toxicity After Dermal Exposure to Hydrofluoric Acid. *J Toxicol Clin Toxicol* 42(4):355-61, 2004.
49. Yamaura, K. et al.: Recurrent Ventricular Tachyarrhythmias Associated with QT Prolongation Following Hydrofluoric Acid Burns. *Clin Toxicol* 35:311-3, 1997.
50. Buckingham, F. M.: Surgery: A Radical Approach to Severe Hydrofluoric Acid Burns. *J Occup Med* 30:873-4, November, 1988.
51. Tsonis, L. et al.: Hydrofluoric Acid Inhalation Injury. *J Burns Care Res* 2008; 29:852-855.
52. Gradinger R. et al.: Toxic Myocarditis Due to Oral Ingestion of Hydrofluoric Acid. *Heart, Lung and Affiliation*, 2008;17:248-250.

Appendix

FIRST AID AND MEDICAL SUPPLIES

The following supplies should be maintained in a dispensary or first aid station near hydrofluoric acid handling and storage areas:

1. Benzalkonium chloride (Zephiran) solution*

- a. For soaks and compresses, 3 to 4 gallons of 0.13% water solution of benzalkonium chloride (Zephiran). The 0.13% solution is available as a non-prescription drug in gallon containers. The solution should be obtained in advance. It should be replaced before the expiration date on the label. It is recommended that it be stored in properly labeled light-resistant containers.

Benzalkonium chloride (Zephiran) is also available as a 17% solution. If this concentrate is used to make a 0.13% (1:750) solution, the dilution should be performed by a qualified individual, such as a registered pharmacist. The shelf life of the diluted solution is uncertain, and it should be replaced annually.

Benzalkonium chloride (Zephiran) should be available as a non-prescription drug through most local pharmacies. The local pharmacies obtain it from pharmaceutical wholesale distributors.

In addition to benzalkonium chloride (Zephiran), benzethonium chloride (Hyamine 1622®) has also been used successfully to treat HF burns. Because of its availability as a nonprescription drug, benzalkonium chloride (Zephiran) is recommended.

- b. Ice cubes (not crushed or shaved ice).
- c. Assorted basins (for immersing burned areas in benzalkonium chloride (Zephiran) solution).
- d. Towels (for use as wet compresses).

2. Calcium gluconate gel, 2.5%

Calcium gluconate gel is available commercially.

It may also be made by mixing one ampule of 10% calcium gluconate solution for each ounce of a water based lubricating jelly (e.g., K-Y® Brand Lubricating Jelly) using 40 cc per 4 ounce tube. This has the advantage that the

ingredients may be readily available. In addition, the ingredients may be stored separately until needed, and shelf life is less of a concern.

In an emergency calcium gluconate gel (2.5% calcium gluconate in a water soluble base) may also be formulated by a pharmacist by dissolving 3.2 grams of calcium gluconate USP in 5 cc of sterile water, and then mixing with 120 cc (4 oz. tube) of K-Y® Jelly or other water soluble lubricant (2.5 grams per 100 cc lubricant).

3. Aqueous calcium gluconate, 10% USP, 10 cc ampules (4.5 mEq calcium or 93 mg elemental calcium per 10 cc)

- a. To make calcium gluconate gel, or
- b. To mix with sterile saline for eye irrigation (5 ampules 10% calcium gluconate per 500 cc sterile normal saline for a 1% solution), or
- c. To mix with sterile saline for administration with oxygen by nebulization (10 cc 10% calcium gluconate in 30 cc sterile saline for a 2.5% solution), or
- d. To be administered by a physician. When injected subcutaneously, 10% calcium gluconate must be diluted 50:50 or 25:75 with normal saline to make 5% or 2.5% solutions.

4. Sterile 0.9% saline

- a. Vials, (e.g. 10 cc, 30 cc, or 50 cc) to dilute 10% calcium gluconate to 2.5% - 5% for injection, or to 2.5% for nebulization.
- b. 500 cc IV to dilute 10% calcium gluconate to 1% for eye irrigation.

5. 0.5% tetracaine hydrochloride solution to counteract blepharospasm and facilitate eye irrigation.

6. Medical oxygen

7. Nebulizer, to administer 2.5% calcium gluconate with oxygen.

8. Beta adrenergic bronchodilators and steroids for inhalation.

9. Surgical gloves

10. Syringes and needles (27-30 gauge).

The FIRST AID AND MEDICAL TREATMENTS AND SUPPLIES recommended in this brochure are based on information reported in the medical literature and the personal experience of Honeywell physicians. It should be noted that there are no medications in the U.S. for which the specific indication is the treatment of HF burns. The physician has the dilemma of using prescription drugs in a non-approved manner, or of using substances which are not approved drugs but which have been proven effective for medical treatment. Given the choice between recommending effective treatment, or recommending the use of only drugs which are approved, we have chosen to recommend the effective treatment.

Benzalkonium chloride (Zephiran) is available in the U.S. as a non-prescription drug. It is a surface active agent sold for use as a disinfectant. It is available in a 1:750 (0.13%) aqueous solution, a 17% concentrate, and a tinted tincture. The concentrated 17% solution must be diluted. The tinted tincture is not recommended to treat HF exposures.

CALCIUM GLUCONATE INJECTION, USP (one gram in 10 ml, 10% solution) is labeled for intravenous use only. Experience has shown that when diluted to 2.5% - 5% with normal saline, and used as described in this brochure, it is a safe and effective treatment for HF skin exposure. When diluted to 2.5% and used as described, it is safe for nebulization and inhalation, and when diluted to 1.0% and used as described, it is safe for eye irrigation.

NOTES

Caltrate® is a Registered Trademark of Wyeth Consumer Healthcare, Madison, NJ 07940

Garamycin® is a Registered Trademark of Fera Pharmaceuticals LLC, Locust Valley, NY 11560

Hyamine® is a Registered Trademark of Rohm and Haas Company, Philadelphia, PA 19106

K-Y® Brand Lubricating Jelly is a Registered Trademark of Johnson & Johnson Products, Inc., Skillman, NJ 08558

Maalox® is a Registered Trademark of McNeil Consumer Pharmaceuticals Co., Fort Washington, PA 19034

Silvadene® is a Registered Trademark of King Pharmaceuticals Research and Development Inc. Cary, NC 27513

Tums® is a Registered Trademark of Glaxo SmithKline Consumer Healthcare, L.P., Pittsburgh, PA 15230

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